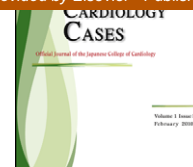




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Case Report

Right ventricular rupture induced by cardiopulmonary resuscitation

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KEYWORDS

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Summary Right ventricular rupture is a rare complication of cardiopulmonary resuscitation and could be fatal. We report a survival case of right ventricular rupture induced by cardiopulmonary resuscitation in a patient with acute myocardial infarction. A 57-year-old man was admitted to our hospital with ventricular fibrillation. Although chest compression and defibrillation were performed, ventricular fibrillation continued. We inserted a percutaneous cardiopulmonary system and performed coronary angiography, which revealed occlusion of the left anterior descending artery. After coronary stenting and intra-aortic balloon pumping, we succeeded in defibrillation and vital signs became stable. Twenty hours after the intervention, systolic blood pressure dropped to 60 mmHg. Ultrasonic cardiogram at that time revealed massive pericardial effusion. We diagnosed cardiac tamponade, and 8Fr drainage tube was placed in the pericardial space. We determined that emergent operation was necessary because we suspected left ventricular rupture due to acute myocardial infarction or coronary rupture induced by percutaneous coronary intervention. However, operative findings revealed right ventricular free wall rupture, which could have been induced by chest compression. In these cases, we should consider the possibility of not only the rupture of left ventricle and coronary artery but also the rupture of right ventricle induced by cardiopulmonary resuscitation.

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Introduction

Right ventricular (RV) rupture is one of the rare complications of cardiopulmonary resuscitation (CPR), and could be fatal. We report a survival case of RV rupture induced by CPR in a patient with acute myocardial infarction (AMI).

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Case report

A 57-year-old man (height: 177 cm; body weight: 61 kg) experienced a ventricular fibrillation (VF) attack in his car while waiting for a traffic signal. An ambulance was called and emergency services arrived about 5 min later. Although chest compression and defibrillation were performed, VF continued. The ambulance arrived at our hospital 20 min after cardiopulmonary arrest. We continued CPR along with the advanced cardiopulmonary life support protocol, but VF persisted. We promptly inserted a percutaneous cardiopulmonary system (PCPS) under the guidance of fluoroscopy (total CPR time was about 35 min) and performed coronary angiography (CAG) which revealed occlusion of the left anterior descending artery (LAD). After successful reperfusion by coronary stenting to the LAD and intra-aortic balloon pumping (IABP), we tried defibrillation again. Then, the rhythm came back to sinus rhythm. Under the support of PCPS, IABP, and intravenous dopamine ($5 \mu\text{g/kg/min}$), vital signs became stable (systolic blood pressure was approximately 100 mmHg). Ultrasonic cardiogram (UCG) just after percutaneous coronary intervention (PCI) revealed wall motion abnormality in the anteroapical–apical area, and slight pericardial effusion. However, systolic blood pressure was gradually decreasing from 15 h after PCI. Twenty hours after PCI, systolic blood pressure dropped to 60 mmHg and heart rate rose to 120 beats/min. UCG at that time revealed massive pericardial effusion, so we diagnosed cardiac tamponade. However, there were not any apparent leak flows. Pericardial effusion could have accumulated gradually after the recovery of hemodynamics. An 8Fr drainage tube was placed in the pericardial space, and 330 ml of bloody fluid was withdrawn. Discharge from the drainage tube continued and approximately 100 ml was withdrawn during the next 1 h. We determined that emergent operation was necessary because we suspected left ventricular oozing rupture due to AML or coronary rupture induced by PCI. However, operative findings revealed subcutaneous and pericardial hematoma induced by CPR and RV free wall rupture (Figs. 1 and 2). No apparent sternum and rib fractures were found. The laceration was about 1.5 cm and placed at the anterior wall of RV. It was sutured by 4-0 PROLENE™ (Ethicon, Inc., Somerville, NJ) (Fig. 3). Two days after the operation, cardiac function ameliorated (cardiac index was 3.6 L/min/m^2 and pulmonary artery wedge pressure was 13 mmHg), and PCPS was removed. IABP and pericardial drainage tube were also removed at post-operative days 3 and 6, respectively.

Discussion

The most frequent complication of CPR is skeletal injury, especially fracture of the rib and sternum. But severe life-threatening complications are rare. Krischer et al. reported the prevalence of life-threatening complications as less than 0.5% [1]. RV rupture is exceedingly rare and fatal [2,3]. The causes of RV rupture are as follows: RV infarction due to right coronary artery occlusion; perforation induced by catheter placed in RV; and trauma. Emergent CAG showed that neither right coronary artery nor RV branch perfusion was hampered at all. Furthermore, neither guide wire nor catheter was inserted into the RV under the guidance of

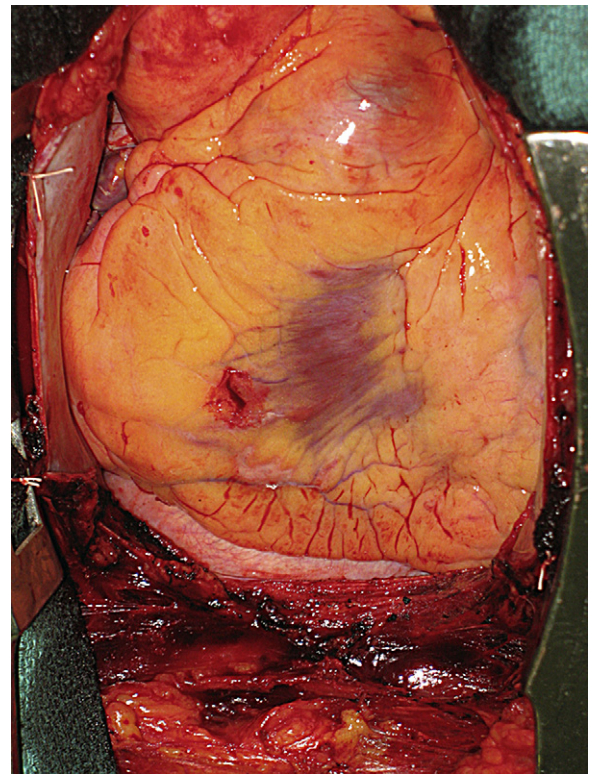


Figure 1 Right ventricular rupture was observed at the operation.

fluoroscopy. The site of RV rupture was under the sternum and placed between the sternum and vertebra. For these reasons, traumatic injury induced by CPR was the most suspicious cause of RV rupture. The causes of cardiac rupture by CPR are reported to be direct compression of the chest, compression between the sternum and vertebral column, injuries induced by sternum or rib fractures [4], and so on. In this case, no apparent fractures were found. Therefore,

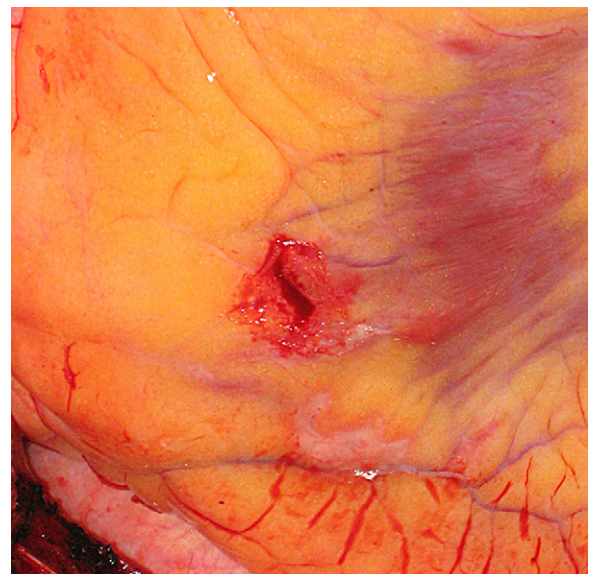


Figure 2 Enlarged image of the laceration.

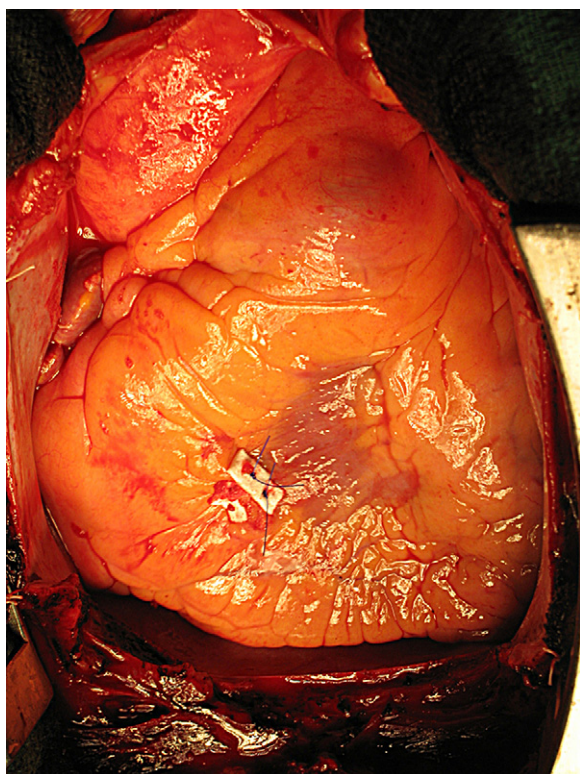


Figure 3 The laceration was sutured.

compression due to CPR might be the cause of RV rupture. The mechanism of RV rupture without fractures might be the result from trapping of blood in the right ventricle at levels of systemic arterial pressure. Massive bilateral pulmonary embolism is reported to be one of the causes of RV rupture induced by chest compression because of reducing the caliber of the pulmonary artery and thereby interfering

with the flow of blood from the right ventricle [5]. In this case, there was no apparent cause for obstruction of RV outflow. We suppose that the position of chest compression was higher than recommended which closed the right ventricular outflow tract, and at the same time, the tricuspid valve was closed by the pressure. This situation might have caused RV rupture. Optimal position of chest compression could have prevented this critically hard complication of CPR.

In AMI cases with acute cardiac tamponade, LV rupture usually comes as the first cause and the next might be coronary rupture due to PCI. There was no apparent extravasation just after PCI, so the coronary rupture was not so suspicious. We considered performing CAG or left ventriculography (LVG) for diagnosis, but we judged that operation should be taken immediately. Operative findings unexpectedly revealed RV rupture as a cause of cardiac tamponade. If we had performed only CAG or LVG before the operation, we could have failed to diagnose and delayed the operation. In these cases, we should consider the possibility of not only the rupture of LV and coronary artery but also the rupture of RV induced by CPR.

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